```
=> d his

     (FILE 'HOME' ENTERED AT 11:53:02 ON 23 SEP 2011)

     FILE 'MEDLINE, CAPLUS, BIOSIS, SCISEARCH, LIFESCI' ENTERED AT 11:53:46 ON
     23 SEP 2011
L1        18076 S (ACTIVAT? OR PROLIFERAT? OR TRANSDUC? OR TRANSFECT?)(6A)(HELP
L2         1987 S (MHC(3A)CLASS(W)I)(7A)(T(W)CELL(W)RECEPTOR OR TCR)
L3           13 S L1(P)L2
L4            6 DUP REM L3 (7 DUPLICATES REMOVED)

=> d au ti so pi 1-6 l4

L4   ANSWER 1 OF 6     MEDLINE on STN                    DUPLICATE 1
AU   Gerner Wilhelm; Kaser Tobias; Saalmuller Armin
TI   Porcine T lymphocytes and NK cells--an update.
SO   Developmental and comparative immunology, (2009 Mar) Vol. 33, No. 3, pp.
     310-20.  Electronic Publication: 2008-07-02. Ref: 92
     Journal code: 7708205. E-ISSN: 1879-0089. L-ISSN: 0145-305X.

L4   ANSWER 2 OF 6  CAPLUS  COPYRIGHT 2011 ACS on STN
AU   Chhabra, Arvind; Yang, Lili; Wang, Pin; Comin-Anduix, Begona; Das, Raja;
     Chakraborty, Nitya G.; Ray, Swagatam; Mehrotra, Shikhar; Yang, Haiguang;
     Hardee, Cinnamon L.; Hollis, Roger; Dorsky, David I.; Koya, Richard; Kohn,
     Donald B.; Ribas, Antoni; Economou, James S.; Baltimore, David; Mukherji,
     Bijay
TI   CD4+CD25- T cells transduced to express MHC class I-restricted
     epitope-specific TCR synthesize Th1 cytokines and exhibit MHC class
     I-restricted cytolytic effector function in a human melanoma model
SO   Journal of Immunology (2008), 181(2), 1063-1070
     CODEN: JOIMA3; ISSN: 0022-1767

L4   ANSWER 3 OF 6  BIOSIS  COPYRIGHT (c) 2011 The Thomson Corporation  on STN
AU   Rohrbach, Florian [Reprint Author]; Brandenburg, Gunda; Ferreira, Edite
     Antunes; Wimmenauer, Vera; Hirsch, Daniel; Stanislawski, Thomas; Huber,
     Christoph; Theobald, Matthias
TI   Phenotype, function, and safety of a p53 TCR bicistronic GMP-suitable
     retroviral construct.
SO   Blood, (NOV 16 2006) Vol. 108, No. 11, Part 1, pp. 1055A-1056A.
     Meeting Info.: 48th Annual Meeting of the American-Society-of-Hematology.
     Orlando, FL, USA. December 09 -12, 2006. Amer Soc Hematol.
     CODEN: BLOOAW. ISSN: 0006-4971.

L4   ANSWER 4 OF 6  BIOSIS  COPYRIGHT (c) 2011 The Thomson Corporation  on STN
     DUPLICATE 2
AU   Morris, E. C. [Reprint Author]; Tsallios, A.; Bendle, G.; Xue, S.; Stauss,
     H. J.
TI   Functional analysis of TCR-transduced MHC class I-restricted
     helper T cells and their role in tumor protection.
SO   Immunology, (DEC 2005) Vol. 116, No. Suppl. 1, pp. 32.
     Meeting Info.: Annual Congress of the British-Society-for-Immunology.
     Harrogate, ENGLAND. December 06 -09, 2005. British Soc Immunol.
     CODEN: IMMUAM. ISSN: 0019-2805.

L4   ANSWER 5 OF 6     MEDLINE on STN                    DUPLICATE 3
AU   Lustgarten J; Waks T; Eshhar Z
TI   CD4 and CD8 accessory molecules function through interactions with major
     histocompatibility complex molecules which are not directly associated
     with the T cell receptor-antigen complex.
SO   European journal of immunology, (1991 Oct) Vol. 21, No. 10, pp. 2507-15.
     Journal code: 1273201. ISSN: 0014-2980. L-ISSN: 0014-2980.
```

L4 ANSWER 6 OF 6 MEDLINE on STN
AU Mukherji B; Chakraborty N G; Sivanandham M
TI T-cell clones that react against autologous human tumors.
SO Immunological reviews, (1990 Aug) Vol. 116, pp. 33-62. Ref: 51
Journal code: 7702118. ISSN: 0105-2896. L-ISSN: 0105-2896.

=> d ab 4-6 l4

L4 ANSWER 4 OF 6 BIOSIS COPYRIGHT (c) 2011 The Thomson Corporation on STN
DUPLICATE 2

L4 ANSWER 5 OF 6 MEDLINE on STN DUPLICATE 3
AB Both the subset-specific, CD4 and CD8 T cell accessory molecules and the antigen-specific T cell receptor (TcR) interact with major histocompatibility complex (MHC) class I and class II molecules on the surface of antigen-presenting cells. We analyzed whether the CD4/CD8 molecules exert their accessory function through binding with the same MHC molecules which participate in the TcR-antigen-MHC complex. We utilized a CD4-, CD8-, class I-allospecific T cell hybridoma which functionally manifests both cytotoxic T lymphocyte (CTL) and T helper1 (Th1) phenotypes, and rendered it bispecific by transfecting it with genes encoding either a class II-restricted, 2,4,6-trinitrophenyl (TNP)-I-Ad-specific TcR or a non-MHC-restricted chimeric TcR, composed of a variable part of an anti-TNP antibody. Expression of either CD4 or CD8 transgenes in these hybridomas enhanced and augmented their reactivity towards the appropriate target cells regardless of the type of TcR-MHC interaction. Thus, class I-specific responses could be enhanced through CD4-class II interactions, and class II-restricted responses could be augmented through CD8-class I interactions. Furthermore, these accessory molecules also potentiated TNP-specific responses by the chimeric TcR which is MHC unrestricted. The accessory molecules facilitated both interleukin 2 (IL2) production and cytolytic activity by shortening the activation time and rendering the cells responsive to lower antigenic stimuli. The degree of activity of the T cell hybridomas correlated with the level of accessory molecule expression and was not related to the effector function mediated by the cells. Anti-CD4 or -CD8 antibodies completely inhibited the activity of transfectants expressing the corresponding accessory molecule, regardless of the MHC type of the TcR interaction. Such antibodies blocked direct TcR stimulation provided by either anti-T3/Ti antibodies or lectins, but could not inhibit the activation through agents that bypass the TcR such as phorbol 12-myristate 13-acetate plus ionophore. Taken together, these studies demonstrate that the CD8/CD4 molecules can exert their accessory function through interactions with MHC molecules which are not directly associated with the TcR-Ag-MHC complex, and that this accessory effect is associated with TcR-mediated triggering at an early stage of the signaling process and is not related to the effector mechanism assigned to the CD4 and CD8 T cell subsets.

L4 ANSWER 6 OF 6 MEDLINE on STN
AB T cells (derived from peripheral blood lymphocytes [PBL], lymph nodes or tumor tissues and restimulated with autologous tumor cells and expanded in interleukin-2 [IL-2]), when cloned, produce three functional classes of clone. Class I T-cell clones exhibit the phenotype of alpha/beta cytotoxic T lymphocytes (CD3+, CD8+, CD4-, WT31+), use their CD3-alpha/beta complexes for cognate function, and lyse the autologous tumor cells specifically in a major histocompatibility complex (MHC) Class I-restricted manner. The second class of T cell clone expresses identical phenotype but exhibits a rather broad cytotoxic profile against the

autologous and allogeneic tumor cells derived from tumors with similar and/or dissimilar histologies. Although these CTL clones can, at times, show MHC Class I-restricted killing and use their T-cell receptors (TCR) complexes for function, activation via certain accessory molecules, particularly lymphocyte-function associated (LFA-1) antigens, might induce their broad cytotoxic behavior. The nature of the tumor antigen recognized by the Class I antigen-specific CTL clones remains unknown. It is evident, however, that more than one antigen can be associated with a given tumor and they are recognized by different CTL clones from individual patients. The third class of T-cell clone is usually of CD4+ alpha/beta T cells (CD3+, CD4+, CD8-, WT31) and these T-cell clones exhibit no cytotoxicity toward the autologous or allogeneic target cells. When tested for potential regulatory property, one type of CD4+ T-cell clone exhibits the characteristics of helper T cells. This type induces or amplifies cytotoxic response in fresh PBL by elaborating interleukin-2 (IL-2) and interferon-gamma). These helper T-cell clones can proliferate against the autologous tumor cells and demonstrate functional specificity for the autologous tumor cells. The other type of CD4+ T-cell clone exhibits the phenotype of the helper T-cell clone (CD3+, CD4+, CD8-, WT31+) but suppresses the cytotoxic response of the autologous PBL in co-culture in the presence of the autologous tumor cells and exogenous IL-2. In some situations, these CD4+ suppressor T-cell clones exhibit considerable specificity for the autologous tumor cells. They do not suppress the cytotoxic response against allogeneic targets or against EBV-infected autologous lymphoblastoid cells. Furthermore, they specifically up-regulate their IL-2 receptors (IL-2R) when stimulated by the autologous tumor cells or with autologous tumor cell-pulsed antigen-presenting cells.(ABSTRACT TRUNCATED AT 400 WORDS)

=>